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Original Research

Depression and Risk for Diabetes: A Meta-Analysis

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ABSTRACT

Objective: Many studies have reported the relationship between depression and diabetes, but the results have been inconsistent. Our aim was to conduct a systematic review through meta-analysis to assess the association of depression with the risk for developing diabetes.
Methods: We retrieved the studies concerning depression and the risk for diabetes. Meta-analysis was applied to calculate the combined effect values and their 95% confidence intervals. The risk for publication bias was assessed by the Egger regression asymmetry test.
Results: As many as 33 articles were included in the meta-analysis, for a total of 2 411 641 participants. The pooled relative risk for diabetes was 1.41 (95% CI, 1.25–1.59) for depression, and the combined relative risk for type 2 diabetes mellitus was 1.32 (95% CI, 1.18–1.47).
Conclusions: Depressed people have a 41% increased risk for developing diabetes mellitus and a 32% increased risk for developing type 2 diabetes. The mechanisms underlying this relationship are still unclear and need further research.

Mots clés :
dépression
diabète
méta-analyse
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RÉSUMÉ

Objectif : De nombreuses études ont rapporté le lien entre la dépression et le diabète, mais les résultats sont apparus contradictoires. Notre objectif était de mener une revue systématique par le recours à la méta-analyse pour évaluer le lien entre la dépression et le risque de développement du diabète.
Méthodes : Nous avons extrait les études concernant la dépression et le risque de diabète. Nous avons eu recours à la méta-analyse pour calculer les valeurs de l'effet combiné et leurs intervalles de confiance à 95 %. Le risque de biais de publication a été évalué à l'aide du test de régression d'Egger.
Résultats : La méta-analyse comportait 33 articles, soit un total de 2 411 641 participants. Lors de dépression, le risque relatif global du diabète était de 1,41 (IC à 95%, 1,25–1,59) et le risque relatif combiné de diabète sucré de type 2 était de 1,32 (IC à 95 %, 1,18–1,47).
Conclusions : Les personnes dépressives montrent une augmentation du risque de développement du diabète sucré de 41 % et une augmentation du risque de développement du diabète de type 2 de 32 %. Les mécanismes sous-jacents à ce lien ne sont pas encore élucidés. D'autres recherches sont nécessaires.

Introduction

Diabetes can damage the heart, blood vessels, eyes, kidneys and nerves, and 50% of people with diabetes die of cardiovascular disease (1). Diabetic retinopathy is an important cause of blindness, and 1% of blindness worldwide can be attributed to diabetes (2). Diabetes is also among the leading causes of kidney failure (3). The overall risk for dying among people with diabetes is at least double the risk of their peers without diabetes (4). There are currently about 347 million people with diabetes worldwide (5). In 2004, an estimated 3.4 million people died from consequences of fasting high blood sugar, and a similar number of deaths has been estimated for 2010 (6). More than 80% of diabetes deaths occur in low- and middle-income countries (7). The World Health Organization (WHO) projects that diabetes will be the seventh leading cause of death in 2030 (3).

The causes of diabetes are complex but are in large part due to rapid increases in overweight, obesity, physical inactivity, sedentary lifestyles and certain dietary behaviours, such as high fat intake

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(8). In addition to these standard risk factors, it has been suggested that depression can increase the risk for diabetes (9,10). Depressive disorders are among the most common of the psychiatric disorders; a recent survey of 38 states in the United States reported the overall prevalence of current depressive symptoms to be 8.7% and found a 15.7% lifetime prevalence rate of diagnosis of a depressive disorder by a doctor or healthcare provider (11). The nature of depression is such that sufferers experience dysphoric mood, loss of interest or pleasure, appetite and sleep disturbances and changes in energy levels. Thus, decreases in selfcare behaviour, such as decreased medication adherence, poor nutrition and lack of exercise, are often associated with depression (12). A number of studies have investigated the relationship between depression and onset of diabetes longitudinally and have shown inconsistent findings. Some report that depression is associated with an increased risk for developing diabetes, whereas other studies do not find a significant association.

The aim of this study was to examine the relationship between depression and the risk for onset of diabetes by conducting a meta-analysis of studies published on this subject in the peer-reviewed literature.

Methods

Literature searches

Studies published in English and Chinese were comprehensively identified in this research. Studies in English were identified through PubMed, MEDLINE, Elsevier Science and Springer Link Cochrane databases from their earliest available dates to March 20, 2013. Chinese articles were screened through China National Knowledge Infrastructure, Database of Chinese Scientific and Technical Periodicals and China biology medical literature databases, which were searched in 1979, 1989, 1970, respectively, through March 21, 2013. The keywords diabetes mellitus or diabetes and depressive disorder or depression or dysthymic disorders or risk factors were used in combination to retrieve the relevant literatures in all these databases. Moreover, the references of all included studies were screened, as were reference lists from reviews and meta-analysis. This systematic review was planned, conducted and reported in adherence to Preferred Reporting Items for Systematic Reviews (PRISMA) guidelines for reporting meta-analyses (13).

Eligibility criteria

Studies were included in the meta-analysis if they met the following criteria: 1) the exposure of interest was depression; 2) the outcome of interest was diabetes; 3) it was a cross-sectional study, case-control study or cohort study; 4) relative risk (RR) or odds ratio (OR) estimates with 95% confidence intervals (CIs) (or data to calculate them) were reported. If data were duplicated in more than 1 study, we included the study with the largest number of cases.

Data extraction

The following data were extracted from each study: first author's last name, publication year, country where the study was performed, study design, range of age, follow up time in years, method of depression assessment, method of diabetes assessment, diabetes type, relative risk and 95% CI (the one adjusted for the largest number of confounders), and adjustment for confounders. Data extraction was conducted independently by 2 authors (Zhang and Lu), with disagreements resolved by consensus.

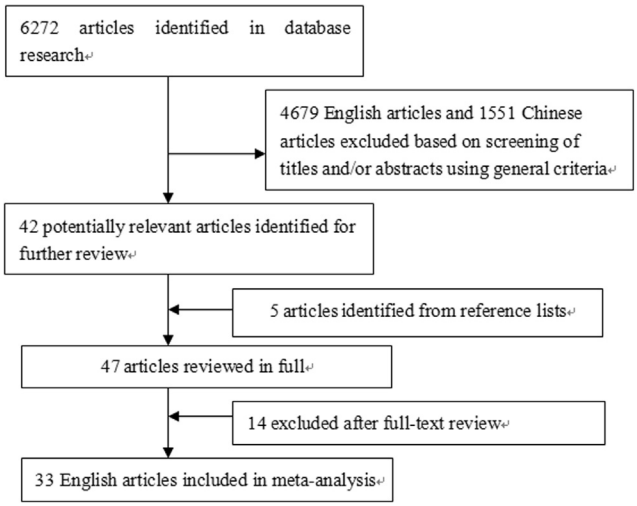

Figure 1. Selection of studies for inclusion in meta-analysis.

Statistical analysis

Data were abstracted from all the studies that met our eligibility criteria. All statistical tests in this study were 2-tailed, and p values of 0.05 or less were considered significant. Statistical analysis was done using Stata, v. 9.2 (StataCorp, College Station, Texas, United States). Estimates of association with diabetes risk were evaluated by RRs and corresponding 95% CIs. Evaluation of meta-analysis results included a test of heterogeneity, sensitivity analyses and examination for bias. Heterogeneity among studies in meta-analysis was assessed by the Cochrane Q statistic, and p values less than 0.10 indicated significant heterogeneity (14). We also used the I^2 statistic to quantify heterogeneity (15). Generally, I^2 values less than 25% correspond to mild heterogeneity; values between 25% and 50% correspond to moderate heterogeneity; and values greater than 50% correspond to large heterogeneity among studies. If the data were heterogeneous, the random effect model was adopted (16), and if the data were homogeneous, the fixed effect model was applied. Sensitivity analyses were done to assess robustness and to examine the results of our meta-analyses for possible bias. Potential publication bias was assessed by using funnel plots of effect sizes vs. standard errors; the Egger regression asymmetry test was used to identify significant asymmetry (17). An analysis of influence was conducted; it describes how robust the pooled estimator is to the removal of individual studies. An individual study is suspected of excessive influence if the point estimate of its omitted analysis lies outside the 95% CI of the combined analysis.

Results

The detailed steps of our literature search are shown in Figure 1. The first approach yielded 6272 publications, which were screened by title and abstract or by a full-text review, if necessary, to identify 47 potentially relevant articles. Two articles were excluded because of a duplicate report based on the same study population, and 12 articles were excluded because of failure to meet the eligibility criteria (8). In the end, 33 publications met our eligibility criteria and were included in the meta-analysis.

The 33 articles (10,18,49) were published between 1991 and 2012 (Table) and involved a total of 2 411 641 participants. Of those, 15 studies were conducted in North America (14 in the United States and 1 in Canada), 13 in Europe, 4 in Asia and 1 in Latin America. The 33 publications included 24 cohort studies, 2 nest case-control studies and 7 cross-sectional studies. Of the articles,